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DATE: Thursday, January 13, 2005

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| ☐ | L4 | L3 and (promoter or gene control region or 5 prime UTR or regulat$ region) | 26 |
| ☐ | L3 | L1 and chick$ | 29 |
| ☐ | L2 | L1 and chicken | 23 |
| ☐ | L1 | intestinal fatty acid binding protein or iFABP or FABP2 | 107 |

END OF SEARCH HISTORY

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        alerts (SDIs) affected
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        alerts (SDIs) affected
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        alerts (SDIs) affected
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        February 2005
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        AND CURRENT DISCOVER FILE IS DATED 10 JANUARY 2005

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=> s intestinal fatty acid binding protein or iFABP or FABP2
L1      1001 INTESTINAL FATTY ACID BINDING PROTEIN OR IFABP OR
FABP2

=> s l1 and (promoter or regulat? region or gene control region or 5 UTR)
L2      82 L1 AND (PROMOTER OR REGULAT? REGION OR GENE CONTROL
REGION OR 5
        UTR)

=> s l2 and chick?
L3      4 L2 AND CHICK?

=> dup rem l3
PROCESSING COMPLETED FOR L3
L4      2 DUP REM L3 (2 DUPLICATES REMOVED)

=> d bib abs 1-
YOU HAVE REQUESTED DATA FROM 2 ANSWERS - CONTINUE? Y/(N):y

L4  ANSWER 1 OF 2  CAPLUS  COPYRIGHT 2005 ACS on STN
AN  2003:737437  CAPLUS
DN  139:256339
TI  Cloning and sequence of a  ***chicken***  I-FABP protein gene, and use
    of the I-FABP  ***promoter***  for gut-specific gene expression in
    transgenic avians and construction of a transgenic bird
IN  Horseman, Nelson D.; Pratt, Scott L.
PA  USA
SO  U.S. Pat. Appl. Publ., 28 pp.
    CODEN: USXXCO
DT  Patent
LA  English
FAN.CNT 1
    PATENT NO.         KIND  DATE      APPLICATION NO.      DATE
    ---------------    ----  --------  -------------------  --------
PI  US 2003177516      A1    20030918  US 2002-99663        20020314
PRAI US 2002-99663           20020314
AB  A recombinant nucleic acid is provided having an avian  ***promoter***  .
    One embodiment of the present invention contemplates the use of a
    gut-specific  ***promoter***  , wherein a  ***promoter***  can be the
     ***chicken***  ***intestinal***  ***fatty***  ***acid***
     ***binding***  ***protein***  (I-FABP)  ***promoter***  region. The
    nucleotide sequence of the  ***chick***  I-FABP gene contg. 1.6 kbp of
    5'-flanking region and the nucleotide sequence of the 0.3 kb I-FABP
    proximal  ***promoter***  -contg. region are provided. A method for
    making a transgenic bird is also disclosed by transfecting a bird with a
    vector comprising a recombinant nucleic acid comprising a  ***chicken***
    I-FABP  ***promoter***  region operably linked to a heterologous nucleic
    acid expressing a desired polypeptide to be expressed in the gut tissue of
    an avian.

L4  ANSWER 2 OF 2  BIOSIS  COPYRIGHT (c) 2005 The Thomson Corporation.
on STN .
    DUPLICATE 1
AN  1992:34170  BIOSIS
DN  PREV199293023445; BA93:23445
TI  SYNTHESIS AND EXPRESSION IN ESCHERICHIA-COLI OF A GENE FOR
KAPPA
    BUNGAROTOXIN.
AU  FIORDALISI J J [Reprint author]; FETTER C H; TENHARMSEL A; GIGOWSKI
R;
    CHIAPPINELLI V A; GRANT G A
CS  DEP BIOCHEMISTRY MOLECULAR BIOPHYSICS, WASHINGTON UNIV
SCHOOL MED, ST
    LOUIS, MISSOURI 63110, USA
SO  Biochemistry, (1991) Vol. 30, No. 42, pp. 10337-10343.
    CODEN: BICHAW. ISSN: 0006-2960.
DT  Article
FS  BA
LA  ENGLISH
ED  Entered STN: 6 Jan 1992
    Last Updated on STN: 6 Mar 1992
AB  A gene which codes for the 66-residue polypeptide of .kappa.-bungarotoxin
    has been chemically synthesized by linking together 3 synthetic
    double-stranded oligonucleotides in a bacterial plasmid. The synthesis
    incoporated six unique silent restriction sites spaced throughout the gene
    for use in cassette mutagenesis. Direct expresssion of the
    .kappa.-bungarotoxin polypeptide by itself in Escherichia coli failed to
    result with rat  ***intestinal***  ***fatty***  ***acid***
     ***binding***  ***protein***  under control of the nalidixic acid
    inducible recA  ***promoter***  . Two fusion protein constructs were
    prepared that differed only in the cleavage site between the fatty acid
    binding protein and the toxin polypeptide. One contained a factor Xa
    cleavage site, and the other, since the toxin itself is devoid of
    methionine, contained a methionyl residue that served as a cyanogen
    bromide cleavage site. The fusion proteins were isolated by ion-exchange
    chromatography and reverse-phase HPLC. The construct containing the
    factor Xa cleavage site could not be cleaved under nondenaturing
    conditions. On the other hand, .kappa.-bungarotoxin was efficiently
    cleaved from the methionyl fusion protein with CNBr. The toxin
    polypeptide was isolated by reverse-phase HPLC and ion-exchange
    chromatography and produced a complete and specific blockade of neuronal
    nicotinic acetylcholine receptors in  ***chick***  ciliray ganglia which
    was indistinguishable from that produced by a comparable amount of
    venom-purified .kappa.-bungarotoxin.


=> dup rem l2
PROCESSING COMPLETED FOR L2
L5      40 DUP REM L2 (42 DUPLICATES REMOVED)

=> d bib abs 1-
YOU HAVE REQUESTED DATA FROM 40 ANSWERS - CONTINUE? Y/(N):y

L5  ANSWER 1 OF 40  CAPLUS  COPYRIGHT 2005 ACS on STN
AN  2004:996011  CAPLUS
DN  142:1361
```

TI Methods of inducing regulated pancreatic hormone production in non-pancreatic islet tissues by increasing PDX-1 expression
IN Ferber, Sarah
PA Israel
SO PCT Int. Appl., 121 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| PI WO 2004098646 | A1 | 20041118 | WO 2004-IB1973 | 20040512 |

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BW, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, EG, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI, NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY, TJ, TM, TN, TR, TT, TZ, UA, UG, UZ, VC, VN, YU, ZA, ZM, ZW
RW: BW, GH, GM, KE, LS, MW, MZ, NA, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRAI US 2003-469715P P 20030512
AB The present application concerns methods of inducing a pancreatic endocrine phenotype and function, including pancreatic hormone prodn., in a non-pancreatic and non-endocrine cell/tissue, particularly in a liver cell/tissue. This is achieved by contacting said non-pancreatic and non-endocrine cell/tissue with a PDX-1 inducer compd., such as a nucleic acid encoding a PDX-1 polypeptide, a neuroD polypeptide or a betacellulin peptide, eventually in the presence of nicotinamide, EGF, activin A, HGF, exendin, GLP-1 or betacellulin.
RE.CNT 8 THERE ARE 8 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 2 OF 40 CAPLUS COPYRIGHT 2005 ACS on STN
AN 2004:1047777 CAPLUS
DN 142:17243
TI Estrogen-related receptor .alpha. (ERR.alpha.) is a transcriptional regulator of apolipoprotein A-IV and controls lipid handling in the intestine
AU Carrier, Julie C.; Deblois, Genevieve; Champigny, Celine; Levy, Emile; Giguere, Vincent
CS Molecular Oncology Group, McGill University Health Center, Montreal, QC, H3A 1A1, Can.
SO Journal of Biological Chemistry (2004), 279(50), 52052-52058
CODEN: JBCHA3; ISSN: 0021-9258
PB American Society for Biochemistry and Molecular Biology
DT Journal
LA English
AB The estrogen-related receptor .alpha. (ERR.alpha.) is an orphan member of the superfamily of nuclear receptors involved in the control of energy metab. In particular, ERR.alpha. induces a high energy expenditure in the presence of the coactivator PGC-1.alpha.. However, ERR.alpha. knockout mice have reduced fat mass and are resistant to diet-induced obesity. ERR.alpha. is expressed in epithelial cells of the small intestine, and because the intestine is the first step in the energy chain, the authors investigated whether ERR.alpha. plays a function in dietary energy handling. Gene expression profiling in the intestine identified a subset of genes involved in oxidative phosphorylation that were down-regulated in the absence of ERR.alpha.. In support of the physiol. role of ERR.alpha. in this pathway, isolated enterocytes from ERR.alpha. knockout mice display lower capacity for .beta.-oxidn. Microarray results also show altered expression of genes involved in dietary lipid digestion and absorption, such as pancreatic lipase-related protein 2 (PLRP2), fatty acid-binding protein 1 and 2 (L-FABP and I-FABP), and apolipoprotein A-IV (apoA-IV). In agreement, the authors found that ERR.alpha.-/- pups exhibit significant lipid malabsorption. The authors further show that the apoA-IV ***promoter*** is a direct target of ERR.alpha. and that its presence is required to maintain basal level but not feeding-induced regulation of the apoA-IV gene in mice. ERR.alpha., in cooperation with PGC-1.alpha., activates the apoA-IV ***promoter*** via interaction with the apoC-III enhancer in both human and mouse. The authors' results demonstrate that apoA-IV is a direct ERR.alpha. target gene and suggest a function for ERR.alpha. in intestinal fat transport, a crucial step in energy balance.
RE.CNT 50 THERE ARE 50 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 3 OF 40 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on STN DUPLICATE 1
AN 2004:235727 BIOSIS
DN PREV200400236220
TI Variation in the ***FABP2*** ***promoter*** affects gene expression: Implications for prior association studies.
AU Formanack, M. L.; Baier, L. J. [Reprint Author]
CS Clinical Diabetes and Nutrition Section, NIDDK, NIH, 4212 North 16th Street, Phoenix, AZ, 85016, USA
lbaier@phx.niddk.nih.gov
SO Diabetologia, (February 2004) Vol. 47, No. 2, pp. 349-351. print.
CODEN: DBTGAJ. ISSN: 0012-186X.
DT Article
LA English
ED Entered STN: 28 Apr 2004
Last Updated on STN: 28 Apr 2004
AB Aims/hypothesis: An Ala54Thr polymorphism in the ***FABP2*** gene has previously been associated with insulin resistance and lipid oxidation rates in Pima Indians. Ala54Thr functionally alters the protein's ability to bind and transport dietary fatty acids. In the current report, we sought additional functional variation in ***FABP2*** by sequencing putative regulatory regions. Methods: More than 1.2 Kb of the putative ***promoter*** of ***FABP2*** was sequenced in 20 Pima subjects. Variations were genotyped in 84 additional Pima Indian subjects to assess haplotype combinations. Functional activities of variant and nonvariant promoters were compared in Caco-2 cells transfected with luciferase reporter constructs. Results: Seven variations were identified in the ***FABP2*** ***promoter*** in Pima Indians. Genotypes of these variants were in complete concordance with each other, and were in complete concordance with Ala54Thr. Therefore, only two ***promoter*** alleles were observed in Pima Indians, an Ala54-associated ***promoter*** and a Thr54-associated ***promoter*** . In contrast, genotyping of these variants in Caucasian DNA showed multiple genotypic combinations. In vitro reporter assays indicated that the Thr54-associated ***promoter*** in Pima Indians resulted in a threefold reduction in ***promoter*** activity as compared to Ala54-associated ***promoter*** . Conclusion/interpretation: Two functional variations exist in ***FABP2*** -the coding Ala54Thr and the variant ***promoter*** . In the Pima Indian population, but not the Caucasian population, these two functional variants are always carried on the same allele. Therefore, some of the in vivo phenotypic associations previously attributed to the Ala54Thr substitution, which alters binding characteristics of the protein, could instead be due to ***promoter*** variation, which alters expression levels.

L5 ANSWER 4 OF 40 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on STN DUPLICATE 2
AN 2004:152705 BIOSIS
DN PREV200400154971
TI Zebrafish ***intestinal*** ***fatty*** ***acid*** ***binding*** ***protein*** (I-FABP) gene ***promoter*** drives gut-specific expression in stable transgenic fish.
AU Her, Guor Mour; Chiang, Chia-Chang; Wu, Jen-Leih [Reprint Author]
CS Institute of Zoology, Academia Sinica, Nankang, Taipei, 115, Taiwan
gmher@gate.sinica.edu.tw; zojlw@ccvax.sinica.edu.tw
SO Genesis The Journal of Genetics and Development, (January 2004) Vol. 38, No. 1, pp. 26-31. print.
ISSN: 1526-954X (ISSN print).
DT Article
LA English
OS DDBJ-AI959004; EMBL-AI959004; GenBank-AI959004
ED Entered STN: 17 Mar 2004
Last Updated on STN: 17 Mar 2004
AB Mammalian ***intestinal*** ***fatty*** ***acid*** ***binding*** ***protein*** (I-FABP) is a small cytosolic protein and is thought to play a crucial role of intracellular fatty acid trafficking and metabolism in gut. To establish an in vivo system for investigating its tissue-specific regulation during zebrafish intestinal development, we isolated 5'-flanking sequences of the zebrafish L-FABP gene and used a transgenic strategy to generate gut-specific transgenic zebrafish with green/red fluorescent intestine. The 4.5-kb 5'-flanking sequence of zebrafish I-FABP gene was sufficient to direct fluorescent expression in intestinal tube, first observed in 3 dpf embryos and then continuously to the adult stage. This pattern of transgenic expression is consistent with the expression pattern of the endogenous gene. In all five transgenic lines 45-52% of the F2 inheritance rates were consistent with the ratio of Mendelian segregation. These fish can also provide a valuable resource of labeled adult intestinal cells for in vivo or in vitro studies. Finally, it is possible to establish an in vivo system using these fish for screening genes required for gut development.

L5 ANSWER 5 OF 40 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on STN
AN 2004:287763 BIOSIS
DN PREV200400286520
TI Positive selection of CD8 alpha-beta+ cells in the thymus permits the generation of CD8 alpha-alpha+ IEL in response to somatic expression of the cognate antigen in the intestine.
AU Perez-Cano, Francisco [Reprint Author]; Hartleroad, Jeffery; Goth, Kirsten; Camerini, Victoria
CS Pediatrics and the Center for Immunology, University of California, Irvine, 843 Health Sciences Court, Irvine, CA, 92697-4120, USA
fperezca@uci.edu
SO FASEB Journal, (2004) Vol. 18, No. 4-5, pp. Abst. 88.5.
http://www.fasebj.org/. e-file.
Meeting Info.: FASEB Meeting on Experimental Biology: Translating the Genome. Washington, District of Columbia, USA. April 17-21, 2004. FASEB.
ISSN: 0892-6638 (ISSN print).
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 16 Jun 2004

Last Updated on STN: 16 Jun 2004
AB Most intestinal intraepithelial lymphocytes (IEL) express CD8 as an alpha-alpha homodimer, a form of CD8 rarely maintained on T cells outside the intestine. While recent studies demonstrate that TL binding to CD8 alpha-alpha may contribute to the functional profile of these IEL, whether encounter with the high affinity ligand in the thymus, intestine or periphery is sufficient for the development of these T cells is unknown. We generated a novel H-Y TCR transgenic mouse line containing the H-2Db restricted cDNA under the ***intestinal*** ***fatty*** ***acid*** ***binding*** ***protein*** ***promoter*** (H-Y Smcy). We found that the thymus of H-Y Smcy female mice contained both CD8+ and CD4+ CD8+ cells similar to the thymus of native H-Y TCR female mice. While T cells in the spleen of H-Y Smcy mice were CD8 alpha-beta+ cells consistent with their positive selection in thymus, the majority of IEL were CD8 alpha-alpha+ cells. As native H-Y TCR female mice have few CD8 alpha-alpha+ IEL, our results suggest that CD8 alpha-beta T cells positively selected in the thymus of H-Y Smcy female mice gave rise to CD8 alpha-alpha + IEL following encounter with the cognate and high affinity ligand in the intestine. We propose that high affinity neo-antigens in the intestine may allow the in situ differentiation of CD8 alpha-alpha+ T cells from conventional CD8 alpha-beta+ T cells thereby broadening the functional profile of CD8+ T cells in the intestine. .

L5 ANSWER 6 OF 40 CAPLUS COPYRIGHT 2005 ACS on STN
AN 2003:757862 CAPLUS
DN 139:274022
TI Transdifferentiation of non-pancreatic cells to pancreatic cells by expressing transcription activation domain VP16 from Herpes simplex virus fusion with pancreatic specific transcription factor
IN Slack, Jonathan; Horb, Marko; Tosh, David
PA The University of Bath, UK
SO PCT Int. Appl., 66 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| PI WO 2003078636 | A1 | 20030925 | WO 2003-GB1180 | 20030317 |
| W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW | | | | |
| RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG | | | | |
| EP 1485485 | A1 | 20041215 | EP 2003-712378 | 20030317 |
| R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO, MK, CY, AL, TR, BG, CZ, EE, HU, SK | | | | |
| PRAI GB 2002-6357 | A | 20020318 | | |
| WO 2003-GB1180 | W | 20030317 | | |

AB The invention relates to transdifferentiation of non-pancreatic cells to pancreatic cells involving the provision of a pancreas specific transcription factor and an activating means such as VP16 of Herpes simplex virus to non-pancreatic cells. The non-pancreatic cells are from liver, lung, thyroid and intestine. The transcription activation domain VP16 from Herpes simplex virus fusion with pancreatic specific transcription factor is under control of tissue specific promoters. The pancreatic transcription factor provided to non-pancreatic cells is the pancreatic transcription factor PDX-1 (mouse) or homologues thereof, such as XlHbox8 (xenopus), STF-1 (rat), IPF-1 (human), or pancreatic transcription factor neurogenin 3 (human). The methods and materials provided by the invention may be used to treat pancreatic disorders, in particular disorders caused by a loss of properly functioning pancreas such as pancreatic cancer and diabetes.
RE.CNT 16 THERE ARE 16 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 7 OF 40 CAPLUS COPYRIGHT 2005 ACS on STN
AN 2003:737437 CAPLUS
DN 139:256339
TI Cloning and sequence of a chicken I-FABP protein gene, and use of the I-FABP ***promoter*** for gut-specific gene expression in transgenic avians and construction of a transgenic bird
IN Horseman, Nelson D.; Pratt, Scott L.
PA USA
SO U.S. Pat. Appl. Publ., 28 pp.
CODEN: USXXCO
DT Patent
LA English
FAN.CNT 1

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| PI US 2003177516 | A1 | 20030918 | US 2002-99663 | 20020314 |
| PRAI US 2002-99663 | | 20020314 | | |

AB A recombinant nucleic acid is provided having an avian ***promoter*** . One embodiment of the present invention contemplates the use of a gut-specific ***promoter*** , wherein a ***promoter*** can be the chicken ***intestinal*** ***fatty*** ***acid*** ***binding*** ***protein*** (I-FABP) ***promoter*** region. The nucleotide sequence of the chick I-FABP gene contg. 1.6 kbp of 5'-flanking region and the nucleotide sequence of the 0.3 kb I-FABP proximal ***promoter*** -contg. region are provided. A method for making a transgenic bird is also disclosed by transfecting a bird with a vector comprising a recombinant nucleic acid comprising a chicken I-FABP ***promoter*** region operably linked to a heterologous nucleic acid expressing a desired polypeptide to be expressed in the gut tissue of an avian.

L5 ANSWER 8 OF 40 CAPLUS COPYRIGHT 2005 ACS on STN
AN 2003:788102 CAPLUS
DN 139:346701
TI A dominant-negative thyroid hormone receptor blocks amphibian metamorphosis by retaining corepressors at target genes
AU Buchholz, Daniel R.; Hsia, Shao-Chung Victor; Fu, Liezhen; Shi, Yun-Bo
CS Unit on Molecular Morphogenesis, Laboratory of Gene Regulation and Development, National Institute for Child Health and Human Development, Bethesda, MD, 20892-5431, USA
SO Molecular and Cellular Biology (2003), 23(19), 6750-6758
CODEN: MCEBD4; ISSN: 0270-7306
PB American Society for Microbiology
DT Journal
LA English
AB The total dependence of amphibian metamorphosis on thyroid hormone (T3) provides a unique vertebrate model for studying the mol. mechanism of T3 receptor (TR) function in vivo. In vitro transcription and developmental expression studies have led to a dual function model for TR in amphibian development, i.e., TRs act as transcriptional repressors in premetamorphic tadpoles and as activators during metamorphosis. We examd. mol. mechanisms of TR action in T3-induced metamorphosis by using dominant-neg. receptors (dnTR) ubiquitously expressed in transgenic Xenopus laevis. We showed that T3-induced activation of T3 target genes and morphol. changes are blocked in dnTR transgenic animals. By using chromatin immunopptn., we show that dnTR bound to target promoters, which led to retention of corepressors and continued histone deacetylation in the presence of T3. These results thus provide direct in vivo evidence for the first time for a mol. mechanism of altering gene expression by a dnTR. The correlation between dnTR-mediated gene repression and inhibition of metamorphosis also supports a key aspect of the dual function model for TR in development: during T3-induced metamorphosis, TR functions as an activator via release of corepressors and promotion of histone acetylation and gene activation.
RE.CNT 76 THERE ARE 76 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 9 OF 40 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on
STN DUPLICATE 3
AN 2003:308761 BIOSIS
DN PREV200300308761
TI Variation in the ***FABP2*** ***promoter*** alters transcriptional activity and is associated with body composition and plasma lipid levels.
AU Damcott, Coleen M. [Reprint Author]; Feingold, Eleanor; Moffett, Susan P.; Barmada, M. Michael; Marshall, Julie A.; Hamman, Richard F.; Ferrell, Robert E.
CS Department of Endocrinology, Diabetes, and Nutrition, University of Maryland School of Medicine, 660 W. Redwood Street, Baltimore, MD, 21201, USA
cdamcott@medicine.umaryland.edu
SO Human Genetics, (May 2003) Vol. 112, No. 5-6, pp. 610-616. print.
CODEN: HUGEDQ. ISSN: 0340-6717.
DT Article
LA English
ED Entered STN: 2 Jul 2003
Last Updated on STN: 2 Jul 2003
AB The fatty acid-binding proteins (FABPs) are cytoplasmic proteins involved in intracellular fatty acid transport and metabolism. ***FABP2*** , the intestinal-type FABP, is expressed exclusively in enterocytes in the small intestine. In previous studies of an Ala54Thr substitution in ***FABP2*** , the Thr-allele showed association with increased lipid oxidation, elevated plasma lipids, and impaired insulin sensitivity. We screened roughly 1 kb 5' of the ***FABP2*** initiation codon and identified three insertion/deletion polymorphisms and four single nucleotide polymorphisms (SNPs). Three of the SNPs were in complete linkage disequilibrium with the three insertion/deletion polymorphisms, defining exactly two haplotypes (FABP2p-ID). We tested the hypothesis that this variation alters gene expression by transfecting Caco-2 cells with pGL3-Basic constructs containing opposite FABP2p-ID haplotypes. Luciferase assays showed a statistically significant two-fold increase in gene expression of the pGL3-insertion construct over the pGL3-deletion construct ($P<0.001$; $n=5$). We also tested for association between three ***FABP2*** variants and measurements of body composition, plasma lipids, and insulin sensitivity in non-diabetic control subjects from the San Luis Valley Diabetes Study ($n=714$). The only informative variant, FABP2p-ID, was statistically significantly associated with body mass index ($P=0.042$) and marginally associated with fat mass ($P=0.084$), cholesterol ($P=0.066$), and HOMA IR (a derived measure of insulin resistance; $P=0.062$) in the entire cohort. Similar associations were seen only in non-Hispanics when the analysis was stratified by ethnicity. Within the non-Hispanic subgroup, the effects of FABP2p-ID on plasma lipids were sex-specific. These results suggest that genetic variation in the 5' region of ***FABP2*** affects transcriptional activity, presumably

leading to alterations in body composition and lipid processing.

L5 ANSWER 10 OF 40 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on
STN
AN 2003:459856 BIOSIS
DN PREV200300459856
TI Variation in the ***FABP2*** ***promoter*** affects gene expression: Implications for prior association studies.
AU Formanack, Mary Lynn [Reprint Author]; Baier, Leslie [Reprint Author]
CS Phoenix, AZ, USA
SO Diabetes, (2003) Vol. 52, No. Supplement 1, pp. A250. print.
Meeting Info.: 63rd Scientific Sessions of the American Diabetes Association. New Orleans, LA, USA. June 13-17, 2003. American Diabetes Association.
ISSN: 0012-1797 (ISSN print).
DT Conference; (Meeting)
Conference; (Meeting Poster)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 8 Oct 2003
Last Updated on STN: 8 Oct 2003

L5 ANSWER 11 OF 40 CAPLUS COPYRIGHT 2005 ACS on STN
AN 2003:91664 CAPLUS
DN 138:349397
TI Genomic structure and expression of a gene coding for a new fatty acid binding protein from Echinococcus granulosus
AU Esteves, Adriana; Portillo, Virginia; Ehrlich, Ricardo
CS Seccion Bioquimica, Facultad de Ciencias, Montevideo, 11400, Urug.
SO Biochimica et Biophysica Acta (2003), 1631(1), 26-34
CODEN: BBACAQ; ISSN: 0006-3002
PB Elsevier Science B.V.
DT Journal
LA English
AB This work describes a new gene coding for a fatty acid binding protein (FABP) in the parasite Echinococcus granulosus, named EgFABP2. The complete gene structure, including the ***promoter*** sequence, is reported. The genomic coding domain organization of the previously reported E. granulosus FABP gene (EgFABP1) has been also detd. The corresponding polypeptide chains share 76% of identical residues and an overall 96% of similarity. The two EgFABPs present the highest amino acid homologies with the mammalian FABP subfamily contg. heart-FABPs (H-FABPs).
The coding sequences of both genes are interrupted by a single intron located in the position of the third intron reported for vertebrate FABP genes. Both genes are expressed in the protoscolex stage of the parasite. The ***promoter*** region of EgFABP2 presents several consensus putative cis-acting elements found in other members of the family, suggesting interesting possible mechanisms involved in the host-parasite adaptation.
RE.CNT 53 THERE ARE 53 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 12 OF 40 CAPLUS COPYRIGHT 2005 ACS on STN
AN 2004:211411 CAPLUS
DN 140:372161
TI Zebrafish ***intestinal*** ***fatty*** ***acid*** ***binding*** ***protein*** (I-FABP) gene ***promoter*** drives gut-specific expression in stable transgenic fish
AU Her, Guor Mour; Chiang, Chia-Chang; Wu, Jen-Leih
CS Laboratory of Marine Molecular Biology and Biotechnology, Institute of Zoology, Academia Sinica, Taipei, Taiwan, Peop. Rep. China
SO Genesis (New York, NY, United States) (2003), Volume Date 2004, 38(1), 26-31
CODEN: GNESFY; ISSN: 1526-954X
PB Wiley-Liss, Inc.
DT Journal
LA English
AB Mammalian ***intestinal*** ***fatty*** ***acid*** - ***binding*** ***protein*** (I-FABP) is a small cytosolic protein and is thought to play a crucial role of intracellular fatty acid trafficking and metab. in gut. To establish an in vivo system for investigating its tissue-specific regulation during zebrafish intestinal development, we isolated 5'-flanking sequences of the zebrafish L-FABP gene and used a transgenic strategy to generate gut-specific transgenic zebrafish with green/red fluorescent intestine. The 4.5-kb 5'-flanking sequence of zebrafish I-FABP gene was sufficient to direct fluorescent expression in intestinal tube, first obsd. in 3 dpf embryos and then continuously to the adult stage. This pattern of transgenic expression is consistent with the expression pattern of the endogenous gene. In all five transgenic lines 45-52% of the F2 inheritance rates were consistent with the ratio of Mendelian segregation. These fish can also provide a valuable resource of labeled adult intestinal cells for in vivo or in vitro studies. Finally, it is possible to establish an in vivo system using these fish for screening genes required for gut development.
RE.CNT 32 THERE ARE 32 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 13 OF 40 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on
STN
AN 2003:391809 BIOSIS
DN PREV200300391809
TI Expression of hCFTR under the control of the ***intestinal*** ***fatty*** ***acid*** - ***binding*** ***protein*** gene ***promoter*** rescues cftr/Clcn3 double knockout mice.
AU Lamb, Fred S. [Reprint Author]; Bailey, Melissa C.; Dickerson, Linda W.; Turner, Jerrold R.; Anders, Robert A.; Schutte, Brian C.
CS Pediatrics, University of Iowa, 200 Hawkins Drive, Iowa City, IA, 52242, USA
fred-lamb@uiowa.edu; melissa-bailey@uiowa.edu; linda-watts-dickerson@uiowa.edu; jturner@bsd.uchicago.edu; randers@bsd.uchicago.edu; brian-schutte@uiowa.edu
SO FASEB Journal, (March 2003) Vol. 17, No. 4-5, pp. Abstract No. 803.13. http://www.fasebj.org/. e-file.
Meeting Info.: FASEB Meeting on Experimental Biology: Translating the Genome. San Diego, CA, USA. April 11-15, 2003. FASEB.
ISSN: 0892-6638 (ISSN print).
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 27 Aug 2003
Last Updated on STN: 27 Aug 2003
AB ClC-3B, a splice variant of the ClC-3 Cl channel, has been hypothesized to be an outwardly rectifying chloride channel (ORCC) that interacts with the cystic fibrosis transmembrane regulator (cftr) (Ogura, FASEB J 16:863). We have previously demonstrated that despite a lack of obvious small intestinal pathology, mice lacking both ClC-3 and cftr do not survive significantly past weaning. We wished to determine if this early demise was related to intestinal epithelial pathology or to the loss of both proteins in some other cell type. Clcn3-/-and cftrtm1Unc double knockout (DKO) mice were created which expressed a human CFTR (hCFTR) transgene under the control of the ***intestinal*** ***fatty*** ***acid*** - ***binding*** ***protein*** gene ***promoter*** . The transgene significantly improved survival (5/5 transgene (-) mice dead by 35 days of age, 8/11 transgene (+) mice surviving >90 days, $p < 0.05$). 90 day old, transgene (+) DKO mice did not display significant lung pathology by light microscopic analysis. These data demonstrate that intestinal epithelial dysfunction is responsible for the death of transgene (-) DKO mice. This finding supports the hypothesis that ClC-3 is an ORCC in the intestinal epithelium and suggests that ClC-3 may contribute to trans-epithelial chloride movement in these cells. Alternatively, the loss of each gene may damage the intestinal epithelium in an independent, but additive fashion.

L5 ANSWER 14 OF 40 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on
STN DUPLICATE 4
AN 2002:185340 BIOSIS
DN PREV200200185340
TI Enterocyte expression of the eotaxin and interleukin-5 transgenes induces compartmentalized dysregulation of eosinophil trafficking.
AU Mishra, Anil; Hogan, Simon P.; Brandt, Eric B.; Wagner, Norbert; Crossman, Michael W.; Foster, Paul S.; Rothenberg, Marc E. [Reprint author]
CS Div. of Allergy and Immunology, Dept. of Pediatrics, Children's Hospital Medical Center, Cincinnati, OH, 45229, USA
rothenberg@chmcc.org
SO Journal of Biological Chemistry, (February 8, 2002) Vol. 277, No. 6, pp. 4406-4412. print.
CODEN: JBCHA3. ISSN: 0021-9258.
DT Article
LA English
ED Entered STN: 6 Mar 2002
Last Updated on STN: 6 Mar 2002
AB Eosinophils accumulate in the gastrointestinal tract in a number of medical disorders, but the mechanisms involved are largely unknown. To understand the significance of cytokine expression by enterocytes, enterocyte transgenic mice that overexpressed the eosinophil-selective cytokines eotaxin and interleukin (IL)-5 were generated. Transgenic mice, generated by utilizing the rat ***intestinal*** ***fatty*** ***acid*** - ***binding*** ***protein*** ***promoter*** (Fabpi), overexpressed the mRNA for these cytokines in the small intestine. Overexpression of IL-5 resulted in marked increases of eosinophils in the bone marrow and blood, whereas eotaxin overexpression resulted in similar levels compared with nontransgenic control mice. In contrast, both IL-5 and eotaxin transgenic mice had significant accumulation of eosinophils in the gastrointestinal mucosa compared with control mice. Eotaxin-induced gastrointestinal eosinophilia was substantially higher than that induced by IL-5 and was especially prominent within the lamina propria of the villi. Interestingly, genetic rescue of eotaxin deficiency (by transgenic overexpression of eotaxin in eotaxin gene-targeted mice) resulted in significant restoration of gastrointestinal eosinophil levels. Finally, the intestinal eosinophilia induced by the eotaxin transgene was beta7 integrin-dependent. Taken together, these results, demonstrate that expression of eotaxin and IL-5 in intestinal epithelium induces compartmentalized dysregulation of eosinophil trafficking and the important role of the beta7 integrin in gastrointestinal allergic responses.

L5 ANSWER 15 OF 40 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on
STN
AN 2002:370834 BIOSIS

DN PREV200200370834
TI Functional autoreactive T cells do not bind MHC I tetramers and modify cytokine responses to unrelated proteins.
AU Vezys, Vaiva [Reprint author]; Marzo, Amanda Lee [Reprint author]; Lefrancois, Leo [Reprint author]
CS Department of Medicine, University of Connecticut Health Center, 263 Farmington Ave, Farmington, CT, 06030, USA
SO FASEB Journal, (March 22, 2002) Vol. 16, No. 5, pp. A1220. print.
Meeting Info.: Annual Meeting of Professional Research Scientists on Experimental Biology. New Orleans, Louisiana, USA. April 20-24, 2002.
CODEN: FAJOEC. ISSN: 0892-6638.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 3 Jul 2002
Last Updated on STN: 3 Jul 2002
AB We have developed a system for exploring antigen-specific tolerance to intestinal antigen. A non-secreted, intracellular form of ovalbumin (ova) was exclusively produced in small intestinal enterocytes of transgenic C57B1/6 mice using the ***intestinal*** ***fatty*** ***acid*** ***binding*** ***protein*** ( ***IFABP*** ) ***promoter*** . We evaluated endogenous CD4 and CD8 T cell responses in these mice using ELISPOT and MHC class I tetramers. Six days post infection with vesicular stomatitis virus expressing ova, no ova-tetramer reactive CD8 T cells were detected, in contrast to non-transgenic controls. However, ELISPOT analysis revealed the presence of both CD4 and CD8 T cells responding to ova in spleen, mesenteric lymph nodes, gut and lung tissues of transgenic mice. These mice exhibited a greater IL-4 response, as compared to controls, which had an increased frequency of interferon-gamma producing T cells. This dichotomy was seen not only for ova epitopes, but for viral epitopes as well. Thus, MHC class I tetramers are not always able to detect functional CD8 T cells and an established response to a self-antigen can impact on subsequent unrelated protein encounters.

L5 ANSWER 16 OF 40 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on
STN DUPLICATE 5
AN 2002:589738 BIOSIS
DN PREV200200589738
TI Aminoglycoside suppression of a premature stop mutation in a Cftr-/- mouse carrying a human CFTR-G542X transgene.
AU Du, Ming; Jones, Julie R.; Lanier, Jessica; Keeling, Kim M.; Lindsey, J. Russell; Tousson, Albert; Bebok, Zsuzsa; Whitsett, Jeffrey A.; Dey, Chitta R.; Colledge, William H.; Evans, Martin J.; Sorscher, Eric J.; Bedwell, David M. [Reprint author]
CS Department of Microbiology, University of Alabama at Birmingham, 1530 Third Avenue, South, Birmingham, AL, 35294-2170, USA
dbedwell@uab.edu
SO Journal of Molecular Medicine (Berlin), (September, 2002) Vol. 80, No. 9, pp. 595-604. print.
ISSN: 0946-2716.
DT Article
LA English
ED Entered STN: 13 Nov 2002
Last Updated on STN: 13 Nov 2002
AB Cystic fibrosis (CF) is caused by mutations in the gene encoding the cystic fibrosis transmembrane conductance regulator (CFTR) protein. Since apprx5% of all mutant CF alleles are stop mutations, it can be calculated that apprx10% of CF patients carry a premature stop mutation in at least one copy of the CFTR gene. Certain ethnic groups, such as the Ashkenazi Jewish population, carry a much higher percentage of CF stop mutations. Consequently, a therapeutic strategy aimed at suppressing this class of mutation would be highly desirable for the treatment of this common genetic disease. We have shown previously that aminoglycoside antibiotics can suppress premature stop mutations in the CFTR gene in a bronchial epithelial cell line (Nat Med (1997) 3:1280). To address whether aminoglycosides can suppress a CFTR premature stop mutation in an animal model, we constructed a transgenic mouse with a null mutation in the endogenous CFTR locus (Cftr-/-) that also expressed a human CFTR-G542X cDNA under control of the ***intestinal*** ***fatty*** ***acid*** ***binding*** ***protein*** ***promoter*** . We then investigated whether the daily administration of the aminoglycoside antibiotics gentamicin or tobramycin could restore the expression of a detectable level of CFTR protein. Immunofluorescence staining of intestinal tissues from Cftr-/- hCFTR-G542X mice revealed that gentamicin treatment resulted in the appearance of hCFTR protein at the apical surface of the glands of treated mice. Weaker staining was also observed in the intestinal glands following tobramycin treatment. Short-circuit current measurements made on intestinal tissues from these mice demonstrated that a significant number of positive cAMP-stimulated transepithelial chloride current measurements could be observed following gentamicin treatment (P=0.008) and a near significant number following tobramycin treatment (P=0.052). When taken together, these results indicate that gentamicin, and to a lesser extent tobramycin, can restore the synthesis of functional hCFTR protein by suppressing the hCFTR-G542X premature stop mutation in vivo.

L5 ANSWER 17 OF 40 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN DUPLICATE 6
AN 2002238842 EMBASE
TI Detection of a ***promoter*** polymorphism in the gene of ***intestinal*** ***fatty*** ***acid*** ***binding*** ***protein*** (I-FABP).
AU Geschonke K.; Klempt M.; Lynch N.; Schreiber S.; Fenselau S.; Schrezenmeir J.
CS M. Klempt, Inst. of Physiol./Biochem. of Nutri., Federal Research Center, Hermann-Weigmann-Strasse 1, 24103 Kiel, Germany. klempt@bafm.de
SO Annals of the New York Academy of Sciences, (2002) 967/- (548-553).
Refs: 18
ISSN: 0077-8923 CODEN: ANYAA
CY United States
DT Journal; Conference Article
FS 003 Endocrinology
022 Human Genetics
029 Clinical Biochemistry
LA English
SL English
AB Postprandial fat absorption is supposed to be a major factor in the development of the metabolic syndrome. In recent years, the assimilation of plasma triglycerides has been the focus of several groups, revealing a number of specific fat or fatty acid transporters. The ***intestinal*** ***fatty*** ***acid*** ***binding*** ***protein*** , I-FABP-2, participates in the absorption of nutritional fats. The influence of a coding polymorphism has been investigated intensively. However, it remains still unclear whether this polymorphism has a major impact on postprandial TG levels in humans. We found a polymorphism in the ***promoter*** of FABP-2, which might involve the retinoid receptor in the transcriptional activity. In functional analysis, we have been able to demonstrate that the various ***promoter*** alleles develop different activities in the human intestinal epithelial cells and that the postprandial appearance of plasma TGs in healthy subjects also depends on their genotype. Since the distribution of the identified ***promoter*** polymorphism does not differ in subjects suffering from type 2 diabetes, the overall influence on the development of the metabolic syndrome seems to be minor.

L5 ANSWER 18 OF 40 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on
STN
AN 2002:508541 BIOSIS
DN PREV200200508541
TI The lac enhancer: An intestinal specific enhancer from the lactase ***promoter*** .
AU Troelsen, Jesper T. [Reprint author]; Olsen, Jorgen [Reprint author]
CS Copenhagen, Denmark
SO Gastroenterology, (April, 2002) Vol. 122, No. 4 Suppl. 1, pp. A.90-A.91. print.
Meeting Info.: Digestive Disease Week and the 103rd Annual Meeting of the American Gastroenterological Association. San Francisco, CA, USA. May 19-22, 2002.
CODEN: GASTAB. ISSN: 0016-5085.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 2 Oct 2002
Last Updated on STN: 2 Oct 2002

L5 ANSWER 19 OF 40 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on
STN DUPLICATE 7
AN 2001:231394 BIOSIS
DN PREV200100231394
TI Down-regulation of beta-catenin TCF signaling is linked to colonic epithelial cell differentiation.
AU Mariadason, John M. [Reprint author]; Bordonaro, Michael; Aslam, Fauzia; Shi, Li; Kuraguchi, Mari; Velcich, Anna; Augenlicht, Leonard H.
CS Department of Oncology, Montefiore Medical Center, Albert Einstein Cancer Center, 111 East 210th Street, Bronx, NY, 10467, USA
john_mariadason@netzero.net
SO Cancer Research, (April 15, 2001) Vol. 61, No. 8, pp. 3465-3471. print.
CODEN: CNREA8. ISSN: 0008-5472.
DT Article
LA English
ED Entered STN: 16 May 2001
Last Updated on STN: 19 Feb 2002
AB The beta-catenin TCF pathway is implicated in the regulation of colonic epithelial cell proliferation, but its role in the regulation of cell differentiation is unknown. The colon carcinoma cell line, Caco-2, spontaneously undergoes G0/G1 cell cycle arrest and differentiates along the absorptive cell lineage over 21 days in culture. In parallel, we show that beta-catenin-TCF activity and complex formation are significantly down-regulated. The down-regulation of beta-catenin-TCF signaling was independent of APC, which we characterized as having a nonsense mutation in codon 1367 in Caco-2 cells, but was associated with a decrease in TCF-4 protein levels. Total beta-catenin levels increased during Caco-2 cell differentiation, although this was attributable to an increase in the membrane, E-cadherin-associated, fraction of beta-catenin. Importantly, down-regulation of beta-catenin-TCF signaling in undifferentiated Caco-2 cells by three different mechanisms, ectopic expression of E-cadherin, wild-type APC, or dominant negative TCF-4, resulted in an increase in the ***promoter*** activities of two genes that are well-established markers of cell differentiation, alkaline phosphatase and ***intestinal*** ***fatty*** ***acid*** ***binding*** ***protein*** . These studies demonstrate, therefore, that in addition to its established role in the regulation of cell proliferation, down-regulation of the

beta-catenin-TCF pathway is associated with the promotion of a more-differentiated phenotype in colonic epithelial cells.

L5 ANSWER 20 OF 40 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on
STN DUPLICATE 8
AN 2001:529026 BIOSIS
DN PREV200100529026
TI MIP-2 secreted by epithelial cells increases neutrophil and lymphocyte recruitment in the mouse intestine.
AU Ohtsuka, Y.; Lee, J.; Stamm, D. S.; Sanderson, I. R. [Reprint author]
CS Department of Adult and Paediatric Gastroenterology, St Bartholomew's and the Royal London School of Medicine and Dentistry, 59 Bartholomew Close, Suite 31, Dominion House, London, EC1A 7BE, UK
i.r.sanderson@mds.qmw.ac.uk
SO Gut, (October, 2001) Vol. 49, No. 4, pp. 526-533. print.
CODEN: GUTTAK. ISSN: 0017-5749.
DT Article
LA English
ED Entered STN: 14 Nov 2001
Last Updated on STN: 23 Feb 2002
AB Background-Invasion of the intestinal mucosa by leucocytes is a characteristic of intestinal inflammation but the role of the epithelium in orchestrating this recruitment has not been examined in vivo. Cultured intestinal epithelial cells secrete a wide variety of chemokines, often in response to agents present in the intestinal lumen. Macrophage inflammatory protein 2 (MIP-2) is a chemokine that attracts neutrophils, and its secretion from intestinal epithelial cells is enhanced by inflamatory stimuli such as interleukin 1beta. We hypothesised that the production of MIP-2 by epithelial cells would increase leucocyte migration into the intestine. Aim-To study the effects of a chemokine secreted from intestinal epithelial cells in vivo. Methods-MIP-2 was expressed in the mouse intestinal epithelium using an epithelial cell specific ***promoter*** from the gene encoding the ***intestinal*** ***fatty*** ***acid*** ***binding*** ***protein*** . The intestines of these transgenic mice were then analysed. Results-Epithelial cells from transgenic mice expressed MIP-2 but wild-type mice did not. Neutrophil recruitment, examined by myeloperoxidase (MPO) staining and total MPO activity per unit weight of intestine, was significantly increased in transgenic mice in both the small intestine and proximal colon, and this was blocked by anti-MIP-2 antibody treatment. Both intraepithelial and lamina propria lymphocytes were also increased in transgenic mice. They showed chemotactic activity to MIP-2 in the Boyden chambers and expressed MIP-2 receptor (CXCR-2) mRNA confirmed by reverse transcription-polymerase chain reaction. Conclusion-These experiments are the first to show a functional role for epithelial chemokines in vivo and reveal an unexpected role for the neutrophil chemokine MIP-2 in controlling mucosal lymphocyte migration.

L5 ANSWER 21 OF 40 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on
STN DUPLICATE 9
AN 2000:278012 BIOSIS
DN PREV200000278012
TI Enterocyte expression of interleukin 7 induces development of gammadelta T cells and Peyer's patches.
AU Laky, Karen; Lefrancois, Leo; Lingenheld, Elizabeth G.; Ishikawa, Hiromichi; Lewis, Julia M.; Olson, Sara; Suzuki, Kenji; Tigelaar, Robert E.; Puddington, Lynn [Reprint author]
CS Dept. of Medicine, University of Connecticut Health Center, 263 Farmington Ave., Farmington, CT, 06030, USA
SO Journal of Experimental Medicine, (May 1, 2000) Vol. 191, No. 9, pp. 1569-1580. print.
CODEN: JEMEAV. ISSN: 0022-1007.
DT Article
LA English
ED Entered STN: 6 Jul 2000
Last Updated on STN: 7 Jan 2002
AB The intestinal mucosa is suggested to support extrathymic T cell development, particularly for T cell receptor (TCR)-gammadelta intraepithelial lymphocytes (IELs). TCR-gammadelta cell development requires interleukin (IL)-7; IL-7-/- or IL-7 receptor-/- mice lack TCR-gammadelta cells. Using the ***intestinal*** ***fatty*** ***acid*** ***binding*** ***protein*** ( ***iFABP*** ) ***promoter*** , we reinstated expression of IL-7 to mature enterocytes of IL-7-/- mice ( ***iFABP*** -IL7). In ***iFABP*** -IL7 mice, TCR-gammadelta IELs were restored, as were cryptopatches and Peyer's patches. TCR-gammadelta cells remained absent from all other tissues. Likewise, T cell development in thymus and B cell maturation in the bone marrow and spleen retained the IL-7-/- phenotype. Thus, IL-7 expression by enterocytes was sufficient for extrathymic development of TCR-gammadelta cells in situ within the intestinal epithelium and was crucial for organization of mucosal lymphoid tissue.

L5 ANSWER 22 OF 40 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on
STN
AN 1999:173639 BIOSIS
DN PREV199900173639
TI B7-1 expression on intestinal epithelium prevents oral tolerance induction.
AU Iqbal, Nuzhat [Reprint author]; Oliver, James R.; McCabe, Robert P.; Elson, Charles O.; Weaver, Casey T.
CS Dep. Pathol., Univ. Ala. at Birmingham, Birmingham, AL 35233, USA
SO FASEB Journal, (March 12, 1999) Vol. 13, No. 4 PART 1, pp. A606. print.
Meeting Info.: Annual Meeting of the Professional Research Scientists for Experimental Biology 99. Washington, D.C., USA. April 17-21, 1999.
CODEN: FAJOEC. ISSN: 0892-6638.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 5 May 1999
Last Updated on STN: 5 May 1999

L5 ANSWER 23 OF 40 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on
STN DUPLICATE 10
AN 1999:486681 BIOSIS
DN PREV199900486681
TI Intestinal overexpression of EGF in transgenic mice enhances adaptation after small bowel resection.
AU Erwin, Christopher R. [Reprint author]; Helmrath, Michael A.; Shin, Cathy E.; Falcone, Richard A., Jr.; Stern, Lawrence E.; Warner, Brad W.
CS Division of Pediatric Surgery, Children's Hospital Medical Center, 3333 Burnet Ave., Cincinnati, OH, 45229-3039, USA
SO American Journal of Physiology, (Sept., 1999) Vol. 277, No. 3 PART 1, pp. G533-G540. print.
CODEN: AJPHAP. ISSN: 0002-9513.
DT Article
LA English
ED Entered STN: 16 Nov 1999
Last Updated on STN: 16 Nov 1999
AB The effect of direct intestinal overexpression of epidermal growth factor (EGF) on postresection adaptation has been investigated by the production of transgenic mouse lines. A murine pro-EGF cDNA construct was produced, and expression of the EGF construct was targeted to the small intestine with the use of the rat ***intestinal*** ***fatty*** ***acid*** - ***binding*** ***protein*** ***promoter*** . An approximately twofold increase in intestinal EGF mRNA and protein was detected in heterozygous mice. No changes in serum EGF levels were noted. Except for a slightly shortened small intestine, no other abnormal phenotype was observed. Intestinal adaptation (increases in body weight, DNA, protein content, villus height, and crypt depth) was markedly enhanced after a 50% proximal small bowel resection in transgenic mice compared with nontransgenic littermates. This transgenic mouse model permits the study of intestinal adaptation and other effects of EGF in the small intestine in a more physiological and directed manner than has been previously possible. These results endorse a direct autocrine/paracrine mechanism for EGF on enterocytes as a means to enhance adaptation.

L5 ANSWER 24 OF 40 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on
STN DUPLICATE 11
AN 2000:51137 BIOSIS
DN PREV200000051137
TI Gut specific expression using mammalian promoters in transgenic Xenopus laevis.
AU Beck, C. W. [Reprint author]; Slack, J. M. W.
CS Developmental Biology Programme, Department of Biology and Biochemistry, University of Bath, Bath, BA2 7AY, UK
SO Mechanisms of Development, (Nov., 1999) Vol. 88, No. 2, pp. 221-227. print.
CODEN: MEDVE6. ISSN: 0925-4773.
DT Article
LA English
ED Entered STN: 3 Feb 2000
Last Updated on STN: 3 Jan 2002
AB The recent development of transgenic methods for the frog Xenopus laevis provides the opportunity to study later developmental events, such as organogenesis, at the molecular level. Our studies have focused on the development of the tadpole gut, where tissue specific promoters have yet to be identified. We have used mammalian promoters, for the genes elastase, pancreatic duodenal homeobox-1, transthyretin, and ***intestinal*** ***fatty*** ***acid*** ***binding*** ***protein*** to drive green fluorescent protein expression in live tadpoles. All of these were shown to drive appropriate tissue specific expression, suggesting that the molecular mechanisms organising the gut are similar in amphibians and mammals. Furthermore, expression from the elastase ***promoter*** is initiated in the pancreatic buds before morphological definition becomes possible, making it a powerful tool for the study of pancreatic determination.

L5 ANSWER 25 OF 40 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on
STN DUPLICATE 12
AN 1998:254033 BIOSIS
DN PREV199800254033
TI Distinct functions are implicated for the GATA-4, -5, and -6 transcription factors in the regulation of intestine epithelial cell differentiation.
AU Gao, Xiaoping; Sedgwick, Tiffany; Shi, Yun-Bo; Evans, Todd [Reprint author]
CS 1300 Morris Park Ave., Chanin 503, Bronx, NY 10461, USA
SO Molecular and Cellular Biology, (May, 1998) Vol. 18, No. 5, pp. 2901-2911. print.
CODEN: MCEBD4. ISSN: 0270-7306.

DT Article
LA English
ED Entered STN: 9 Jun 1998
Last Updated on STN: 9 Jun 1998
AB Based on conserved expression patterns, three members of the GATA family of transcriptional regulatory proteins, GATA-4, -5, and -6, are thought to be involved in the regulation of cardiogenesis and gut development. Functions for these factors are known in the heart, but relatively little is understood regarding their possible roles in the regulation of gut-specific gene expression. In this study, we analyze the expression and function of GATA-4, -5, and -6 using three separate but complementary vertebrate systems, and the results support a function for these proteins in regulating the terminal-differentiation program of intestinal epithelial cells. We show that xGATA-4, -5, and -6 can stimulate directly activity of the ***promoter*** for the ***intestinal*** ***fatty*** ***acid*** - ***binding*** ***protein*** (xIFABP) gene, which is a marker for differentiated enterocytes. This is the first direct demonstration of a target for GATA factors in the vertebrate intestinal epithelium. Transactivation by xGATA-4, -5, and -6 is mediated at least in part by a defined proximal ***IFABP*** ***promoter*** element. The expression patterns for cGATA-4, -5, and -6 are markedly distinct along the proximal-distal villus axis. Transcript levels for cGATA-4 increase along the axis toward the villus tip; likewise, cGATA-5 transcripts are largely restricted to the distal tip containing differentiated cells. In contrast, the pattern of cGATA-6 transcripts is complementary to cGATA-5, with highest levels detected in the region of proliferating progenitor cells. Undifferentiated and proliferating human HT-29 cells express hGATA-6 but not hGATA-4 or hGATA-5. Upon stimulation to differentiate, the transcript levels for hGATA-5 increase, and this occurs prior to increased transcription of the terminal differentiation marker intestinal alkaline phosphatase. At the same time, hGATA-6 steady-state transcript levels decline appreciably. All of the data are consistent with evolutionarily conserved but distinct roles for these factors in regulating the differentiation program of intestinal epithelium. Based on this data, we suggest that GATA-6 might function primarily within the proliferating progenitor population, while GATA-4 and GATA-5 function during differentiation to activate terminal-differentiation genes including ***IFABP*** .

L5 ANSWER 26 OF 40 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on
STN
AN 1998:285592 BIOSIS
DN PREV199800285592
TI Adenovirus mediated enterocyte specific expression of a beta-galactosidase/green fluorescence protein chimeric reporter using an ***intestinal*** ***fatty*** ***acid*** ***binding*** ***protein*** ***promoter*** .
AU Li, Wei [Reprint author]; Rosenzweig, Anthony; Grand, Richard J.
CS Div. Pediatr. Gastroenterol. and Nutrition, GRASP Ctr., Boston, MA, USA
SO Gastroenterology, (April 15, 1998) Vol. 114, No. 4 PART 2, pp. A391. print.
Meeting Info.: Digestive Disease Week and the 99th Annual Meeting of the American Gastroenterological Association. New Orleans, Louisiana, USA. May 16-22, 1998. American Gastroenterological Association.
CODEN: GASTAB. ISSN: 0016-5085.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 8 Jul 1998
Last Updated on STN: 8 Jul 1998

L5 ANSWER 27 OF 40 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN
AN 1998076386 EMBASE
TI Nutrient absorption.
AU Kumar N.S.; Nutting D.F.; Hilaire J.St.; Mansbach C.M.
CS Dr. N.S. Kumar, The University Tennessee, Memphis, 951 Court Avenue, Memphis, TN 38163, United States
SO Current Opinion in Gastroenterology, (1998) 14/2 (99-106).
Refs: 35
ISSN: 0267-1379 CODEN: COGAEK
CY United States
DT Journal; General Review
FS 002 Physiology
005 General Pathology and Pathological Anatomy
048 Gastroenterology
LA English
SL English
AB Considerable advances occurred during the past year in nutrient absorption. The movement of triacylglycerol from the endoplasmic reticulum to the Golgi was studied with the finding that it is likely a vesicle-based cytosolic protein dependent on ATP and that the delivery is specific for intestinal Golgi. With respect to gene regulation, important information concerning the editing of the full length apolipoprotein B transcript was generated, including the identification of two promoters that are tissue specific and a polymorphism in the human gene that produces a frame shift resulting in a truncated, inactive apobec-1 peptide. ***Intestinal*** ***fatty*** ***acid*** ***binding*** ***protein*** was shown to be induced by peptide YY. The cystic fibrosis transmembrane regulator was shown to be more closely linked to the Na+-glucose cotransporter than expected. Mutations in the Na+-glucose and ileal Na+-bile acid cotransporter were shown to account for congenital glucose/galactose and bile acid malabsorption, respectively.

L5 ANSWER 28 OF 40 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED.
on STN DUPLICATE 13
AN 1998298202 EMBASE
TI Obtention of porcine aminopeptidase-N transgenic mice and analysis of their susceptibility to transmissible gastroenteritis virus.
AU Benbacer L.; Stinackre M.-G.; Laude H.; Delmas B.
CS L. Benbacer, Unite Virol./Immunol. Moleculaires, Inst. National Recherche Agronomique, F-78350 Jouy-en-Josas, France
SO Advances in Experimental Medicine and Biology, (1998) 440/- (53-59).
Refs: 64
ISSN: 0065-2598 CODEN: AEMBAP
CY United States
DT Journal; Article
FS 004 Microbiology
LA English
SL English
AB To obtain a laboratory animal model for transmissible gastroenteritis virus (TGEV) infection, transgenic mice (Tg) were produced by introducing two porcine aminopeptidase-n (APN) cDNA-derived constructs into the mouse genome. In the first construct, the APN cDNA was fused in 5' with the 1kb upstream region of the APN gene and in 3' with the SV40 small intron and polyadenylation site. In the second construct, the 5' end of the APN cDNA was replaced by the corresponding domain of the APN gene comprising the three first introns, an additional intron (the rabbit .beta.-like globine intron 2) was inserted at the 3' extremity of the construct and the resulting DNA stretch was placed under the control of the rat ***intestinal*** ***fatty*** ***acid*** - ***binding*** ***protein*** (I-FABP) gene ***promoter*** . Transgenes were obtained with these two constructs, and RNA expression was evidenced by RT-PCR with the second construct in a transgene lineage. Using two different immunoassays, expression of the porcine APN protein was not detected in the transgenic intestines of animals of the RT-PCR positive lineage. Northern blot analyses did not revealed TGEV replication in infected adult mice. Additional assays will be carried out on young animals to detect potential TGEV susceptibility.

L5 ANSWER 29 OF 40 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on
STN DUPLICATE 14
AN 1998:47737 BIOSIS
DN PREV199800047737
TI Common genomic variation in the APOC3 ***promoter*** associated with variation in plasma lipoproteins.
AU Hegele, Robert A. [Reprint author]; Connelly, Philip W.; Hanley, Anthony J. G.; Sun, Fang; Harris, Stewart B.; Zinman, Bernard
CS Blackburn Cardiovasc. Genet. Lab., Robarts Res. Inst., Room 406-100, Perth Dr., London, ON N6A 5K8, Canada
SO Arteriosclerosis Thrombosis and Vascular Biology, (Nov., 1997) Vol. 17, No. 11, pp. 2753-2758. print.
ISSN: 1079-5642.
DT Article
LA English
ED Entered STN: 27 Jan 1998
Last Updated on STN: 27 Jan 1998
AB We hypothesized that common genomic variation that affected the expression and/or function of the products of the APOC3, APOE, ***FABP2*** , and PON1 genes would be associated with variation in biochemical phenotypes in a previously unstudied human sample. We determined genotypes of functional genomic variants of APOC3, APOE, ***FABP2*** , and PON1 in 509 adult aboriginal Canadians from an isolated community in Northern Ontario. We tested for genotype associations with plasma lipoprotein traits. We found that (1) common variation at nucleotide -455 of the APOC3 ***promoter*** was associated with variation in plasma triglycerides (P=.006) and (2) common variation of APOE determining plasma isoforms of apo E was associated with variation in plasma apo B (P=.009). Analysis of subjects classed by APOC3 markers showed that homozygosity for presence of a C at nucleotide -455 and a T at nucleotide -482 was associated with significantly increased plasma triglycerides in both men and women. Furthermore, this allele was approximately twice as frequent in subjects within the highest quartile of plasma triglycerides as in subjects within the lowest quartile. Since the DNA variation detected by the APOC3 markers affects in vitro expression of the gene product, it is possible that the marker itself caused the associations. However, the associations could also have resulted from linkage disequilibrium with other functional variants in APOC3 or the closely linked APOA1 and/or APOA4 genes.

L5 ANSWER 30 OF 40 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on
STN DUPLICATE 15
AN 1997:201046 BIOSIS
DN PREV199799500249
TI Metallothionein-II-A ***promoter*** induction alters rat ***intestinal*** ***fatty*** ***acid*** ***binding*** ***protein*** expression, fatty acid uptake, and lipid metabolism in transfected L-cells.
AU Prows, Daniel R.; Schroeder, Friedhelm [Reprint author]
CS Dep. Physiol. Pharmacol., Texas A and M University, TVMC, College Station,

TX 77843-4466, USA
SO Archives of Biochemistry and Biophysics, (1997) Vol. 340, No. 1, pp. 135-143.
CODEN: ABBIA4. ISSN: 0003-9861.
DT Article
LA English
ED Entered STN: 12 May 1997
Last Updated on STN: 12 May 1997
AB Mouse L-cell fibroblasts, transfected with the cDNA encoding for rat ***intestinal*** ***fatty*** ***acid*** - ***binding*** ***protein*** (I-FABP) under the control of the human metallothionein-II-A ***promoter*** , were tested for their protein inducibility by the heavy metals cadmium (Cd-2+) and zinc (Zn-2+). I-FABP leves were quantitated by Western immunoblotting. Expression of I-FABP in all transfected cell lines tested was induced several-fold by optimized levels of Cd-2+ and Zn-2+. Induction conditions had no effect on cell growth rates or cell densities for any of the cell lines. Induction of high I-FABP-expressing cells (H141) decreased the initial rate and extent of uptake of cis-parinaric acid, a nonmetabolizable fatty acid, and of (3H)oleic acid, an esterifiable fatty acid. These effects of induction were specific for I-FABP expressing cells since they were not observed in control cells or cells expressing a high level of liver (L-) FABP. Induction of H141 cells also significantly altered the esterification and distribution of exogenous (3H)oleic acid, especially among triglycerides and phosphatidylcholine, but less so among other glycerophospholipids, cholesteryl esters, and phosphatidylethanolamine. Induction of H141 cells normalized (3H)oleic acid esterification into cholesteryl esters, phosphatidylcholine, total neutral lipids, and total phospholipids such that they no longer differed from control levels. In contrast, induction did not normalize (3H)oleic acid esterification into triacylglycerols and phosphatidylethanolamine a control levels in H141 cells; both remained significantly increased over control cells. Therefore, ***promoter*** induction levels of Cd-2+ and Zn-2+ enhanced I-FABP expression in H141 cells, thereby modulating both fatty acid uptake and intracellular esterification into neutral and phospholipids.

L5 ANSWER 31 OF 40 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on STN DUPLICATE 16
AN 1996:572748 BIOSIS
DN PREV199799287429
TI Use of normal and transgenic mice to examine the relationship between terminal differentiation of intestinal epithelial cells and accumulation of their cell cycle regulators.
AU Chandrasekaran, Chitra [Reprint author]; Coopersmith, Craig M.; Gordon, Jeffrey L. [Reprint author]
CS Dep. Molecular Biol. Pharmacol. Washington Univ. Sch. Med., St. Louis, MO 63110, USA
SO Journal of Biological Chemistry, (1996) Vol. 271, No. 45, pp. 28414-28421.
CODEN: JBCHA3. ISSN: 0021-9258.
DT Article
LA English
ED Entered STN: 23 Dec 1996
Last Updated on STN: 23 Dec 1996
AB A spatially well organized continuum of proliferation, differentiation, and death is displayed along crypt-villus units in the adult mouse small intestine. This continuum provides an opportunity to examine in vivo the mechanisms by which proliferative status changes as a function of cellular differentiation. Immunohistochemical studies of normal FVB/N mice revealed that as epithelial cells complete their terminal differentiation during a 48-72-h migration up villi, there is a marked and rapid fall in the levels of two important regulators of the G-1/S transition, cyclin D-1 and cyclin-dependent kinase (cdk) 2. However, cellular levels of their partners, cdk4 and cyclin E, remain unchanged as does the level of pRB. Adult FVB/N transgenic mice were studied that contained an ***intestinal*** ***fatty*** ***acid*** ***binding*** ***protein*** gene ***promoter*** (Fabpi) linked to wild type Simian virus 40 large T antigen (SV40 TAg-Wt) or a mutant TAg with Lys for Glu substitutions at residues 107 and 108 (SV40 TAg-K107/8) that fails to bind pRB and related pocket proteins. Both transgenes are expressed only in villus enterocytes. SV40 TAg-Wt causes these terminally differentiated cells to re-enter the cycle. Re-entry is accompanied by a reduction in un/hypophosphorylated pRB, an induction of cyclin D-1 and cdk2, but no change in cdk4, cyclin E, or E2F-1. In contrast, SV40 TAg-K107/8 fails to induce reentry and does not produce changes in un/hypophosphorylated pRB, cyclin D-1, or cdk2 accumulation. These results suggest that un/hypophosphorylated pRB is an important mediator of the cell cycle arrest that normally occurs as enterocytes exit the crypt and complete their differentiation. Fabpi-directed expression of E2F-1 does not cause villus enterocytes to return to the cell cycle, alter their suppression of cyclin D-1, or cdk2, or affect their state of differentiation, emphasizing the insensitivity of these cells to the effects of E2F-1. Analyses of p53-/- and p53+/+ mice containing Fabpi-SV40 TAg-Wt and Fabpi-SV40 TAg-K107/8 established that the proliferation induced by SV40 TAg-Wt does not require p53 and is associated with increased (p53-independent) apoptosis. The presence of cyclin E and cdk4 in differentiating villus enterocytes emphasizes that these cells retain part of their proliferative heritage expressed 24-72 h earlier in the crypt. The data suggest that down-regulation of cdk2 and/or cyclin D-1 expression may be important for control of proliferative status and/or execution of terminal differentiation.

L5 ANSWER 32 OF 40 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on STN DUPLICATE 17
AN 1996:537452 BIOSIS
DN PREV199699259808
TI The transcriptional activator hepatocyte nuclear factor 6 regulates liver gene expression.
AU Samadani, Uzma; Costa, Robert H. [Reprint author]
CS Dep. Biochem., Univ. Ill. at Chicago, Coll. Medicine, 1819 W. Polk St., Chicago, IL 60612-7334, USA
SO Molecular and Cellular Biology, (1996) Vol. 16, No. 11, pp. 6273-6284.
CODEN: MCEBD4. ISSN: 0270-7306.
DT Article
LA English
ED Entered STN: 10 Dec 1996
Last Updated on STN: 10 Dec 1996
AB The hepatocyte nuclear factor 3-alpha (HNF-3-alpha), -3-beta, and -3-gamma proteins share homology in the winged-helix/fork head DNA binding domain and mediate hepatocyte-enriched transcription of numerous genes whose expression is necessary for organ function. In this work, we identify a liver-enriched transcription factor, HNF-6, which recognizes the -138 to -126 region of the HNF-3-beta ***promoter*** and binds the original HNF-3 site of the transthyretin ***promoter*** (-94 to -106). We show that HNF-6 and HNF-3 possess different DNA binding specificities by competition and methylation interference studies and are immunologically distinct. Site-directed mutagenesis of the HNF-6 sites in the HNF-3-beta and transthyretin promoters diminishes reporter gene expression, suggesting that HNF-6 activates transcription of these promoters. Using the HNF-6 binding sequence DHWATTGAYTWWD (where W = A or T, Y = T or C, H is not G, and D is not C) determined by sequence comparison and methylation interference, we predicted that HNF-6 will bind to 22 additional hepatocyte-enriched genes. Of these potential target genes, we selected seven of the HNF-6 binding sequences and demonstrated that they bind the HNF-6 protein. These include ***promoter*** sequences from alpha-2 urinary globulin, alpha-1 antitrypsin, cytochrome P-450 2C13, L-type 6-phosphofructo-2-kinase, mouse major urinary protein, tryptophan oxygenase, and alpha-fetoprotein genes. HNF-6 binding activity was also found in the intestinal epithelial cell line HT29, and potential HNF-6 binding sites were present in intestinal sucrase isomaltase, cdx-2 homeodomain protein, and ***intestinal*** ***fatty*** ***acid*** ***binding*** ***protein*** ***promoter*** regions. These studies suggest that HNF-6 may regulate hepatocyte-specific genes and may play a role in epithelial cell differentiation of gut endoderm via regulation of HNF-3-beta.

L5 ANSWER 33 OF 40 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on STN DUPLICATE 18
AN 1996:184726 BIOSIS
DN PREV199698740855
TI Transgenic mice that overexpress the human trefoil peptide pS2 have an increased resistance to intestinal damage.
AU Playford, R. J. [Reprint author]; Marchbank, T.; Goodlad, R. A.; Chinery, R. A.; Poulsom, R.; Hanby, A. M.; Wright, N. A.
CS Dep. Gastroenterol., Leicester General Hosp., Gwendolen Rd., Leicester LE5 4PW, UK
SO Proceedings of the National Academy of Sciences of the United States of America, (1996) Vol. 93, No. 5, pp. 2137-2142.
CODEN: PNASA6. ISSN: 0027-8424.
DT Article
LA English
ED Entered STN: 29 Apr 1996
Last Updated on STN: 29 Apr 1996
AB pS2 is a member of the trefoil peptide family, all of which are overexpressed at sites of gastrointestinal injury. We hypothesized that they are important in stimulating mucosal repair. To test this idea, we have produced a transgenic mice strain that expresses human pS2 (hpS2) specifically within the jejunum and examined the effect of this overexpression on proliferation and susceptibility to indomethacin-induced damage. A transgenic mouse was produced by microinjecting fertilized oocytes with a 1.7-kb construct consisting of rat ***intestinal*** ***fatty*** ***acid*** ***binding*** ***protein*** ***promoter*** (positions -1178 to +28) linked to full-length (490 hp) hpS2 cDNA. Screening for positive animals was by Southern blot analysis. Distribution of hpS2 expression was determined by using Northern and Western blot analyses and immunohistochemical staining. Proliferation of the intestinal mucosa was determined by assessing the crypt cell production rate. Differences in susceptibility to intestinal damage were analyzed in animals that had received indomethacin (85 mg/kg s.c.) 0-30 h previously. Expression of hpS2 was limited to the enterocytes of the villi within the jejunum. In the nondamaged intestine, villus height and crypt cell production rate were similar in transgenic and negative (control) litter mates. However, there was a marked difference in the amount of damage caused by indomethacin in control and transgenic animals in the jejunum (30% reduction in villus height in controls vs. 12% reduction in transgenic animals, P lt 0.01) but the damage sustained in the non-hpS2-expressing ileal region was similar in control and transgenic animals. These studies support the hypothesis that trefoil peptides are important in stimulating gastrointestinal repair.

L5 ANSWER 34 OF 40 CAPLUS COPYRIGHT 2005 ACS on STN
AN 1995:708572 CAPLUS

DN 123:104357
TI Enhanced transgene expression in specific tissues of the gastrointestinal tract by using a tissue-specific ***promoter***
IN Simonet, William S.; Ratzkin, Barry J.
PA Amgen Inc., USA
SO PCT Int. Appl., 56 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

| PATENT NO. | KIND | DATE | APPLICATION NO. | DATE |
|---|---|---|---|---|
| PI WO 9511299 | A1 | 19950427 | WO 1994-US11716 | 19941013 |
| W: AU, CA, JP | | | | |
| RW: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE | | | | |
| CA 2183553 | AA | 19950427 | CA 1994-2183553 | 19941013 |
| AU 9510825 | A1 | 19950508 | AU 1995-10825 | 19941013 |
| EP 733105 | A1 | 19960925 | EP 1995-901685 | 19941013 |
| R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IE, IT, LI, LU, MC, NL, PT, SE | | | | |
| PRAI US 1993-141323 | A | 19931018 | | |
| WO 1994-US11716 | W | 19941013 | | |

AB This invention provides a mammal with enhanced expression of a transgene in certain tissues of the gastrointestinal tract. Also provided are (1) a nucleic acid sequence useful in enhancing expression of a transgene in certain gastrointestinal tissues, and (2) a vector contg. this nucleic acid sequence. The transgene is operably linked to a ***promoter*** selected from the group consisting of the ***intestinal*** ***fatty*** ***acid*** - ***binding*** ***protein*** (FABP) ***promoter*** , liver FABP ***promoter*** , and apolipoprotein C-III ***promoter*** . Thus, PCR-amplified 1210-nucleotide portion of rat intestinal FABP ***promoter*** is inserted into the EcoRI site of pUC19 to generate plasmid FABPTB. Then intron 1 of the apolipoprotein E gene (also contg. portions of its 3' and 5' exons) are inserted into KpnI-cut FABPTB. This construct was then linked to cDNA fragments encoding human interleukin-8 or human keratinocyte growth factor, along with the SV40 polyadenylation sequence. Microinjection of the vector into mouse embryos yielded transgenic mice that displayed serum levels of interleukin-8 of 5-15 mg/mL blood, whereas no interleukin-8 was detectable in non-transgenic control mice.

L5 ANSWER 35 OF 40 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on
STN DUPLICATE 19
AN 1995:510427 BIOSIS
DN PREV199598515477
TI A 20-nucleotide element in the ***intestinal*** ***fatty*** ***acid*** ***binding*** ***protein*** gene modulates its cell lineage-specific, differentiation-dependent, and cephalocaudal patterns of expression in transgenic mice.
AU Simon, Theodore C.; Roberts, Lisa J. J.; Gordon, Jeffrey I. [Reprint author]
CS Dep. Mol. Biol. Pharmacol., Washington Univ. Sch. Med., St. Louis, MO 63110, USA
SO Proceedings of the National Academy of Sciences of the United States of America, (1995) Vol. 92, No. 19, pp. 8685-8689.
CODEN: PNASA6. ISSN: 0027-8424.
DT Article
LA English
ED Entered STN: 29 Nov 1995
Last Updated on STN: 29 Nov 1995
AB A sequence of epithelial cell proliferation, allocation to four principal lineages, migration-associated differentiation, and cell loss occurs along the crypt-villus axis of the mouse intestine. The sequence is completed in a few days and is recapitulated throughout the life-span of the animal. We have used an intestine-specific fatty acid binding protein gene, Fabpi, as a model for studying regulation of gene expression in this unique developmental system. ***Promoter*** mapping studies in transgenic mice identified a 20-bp cisacting element (5'-AGGTGGAAGCCATCACACTT-3') that binds small intestinal nuclear proteins and participates in the control of Fabpi's cephalocaudal, differentiation-dependent, and cell lineage-specific patterns of expression. Immunocytochemical studies using confocal and electron microscopy indicate that it does so by acting as a suppressor of gene expression in the distal small intestine/colon, as a suppressor of gene activation in proliferating and nonproliferating cells located in the crypts of Lieberkuhn, and as a suppressor of expression in the growth factor and defensin-producing Paneth cell lineage. The 20-bp domain has no obvious sequence similarities to known transcription factor binding sites. The three functions modulated by this compact element represent the types of functions required to establish and maintain the intestine's remarkably complex spatial patterns of gene expression. The transgenes described in this report also appear to be useful in characterizing the crypt's stem cell hierarchy.

L5 ANSWER 36 OF 40 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on
STN DUPLICATE 20
AN 1995:63456 BIOSIS
DN PREV199598077756
TI Correction of lethal intestinal defect in a mouse model of cystic fibrosis by human CFTR.
AU Zhou, Lan; Dey, Chitta R.; Wert, Susan E.; Duvall, Michael D.; Frizzell, Raymond A.; Whitsett, Jeffrey A. [Reprint author]
CS Children's Hosp. Med. Cent., Div. Pulmonary Biol., Cincinnati, OH 45229-3039, USA
SO Science (Washington D C), (1994) Vol. 266, No. 5191, pp. 1705-1708.
CODEN: SCIEAS. ISSN: 0036-8075.
DT Article
LA English
ED Entered STN: 8 Feb 1995
Last Updated on STN: 9 Feb 1995
AB Cystic fibrosis (CF) is caused by mutations in the gene encoding the cystic fibrosis transmembrane conductance regulator (CFTR). A potential animal model of CF, the CFTR-/- mouse, has had limited utility because most mice die from intestinal obstruction during the first month of life. Human CFTR (hCFTR) was expressed in CFTR-/- mice under the control of the rat ***intestinal*** ***fatty*** ***acid*** - ***binding*** ***protein*** gene ***promoter*** . The mice survived and showed functional correction of ileal goblet cell and crypt cell hyperplasia and cyclic adenosine monophosphate-stimulated chloride secretion. These results support the concept that transfer of the hCFTR gene may be a useful strategy for correcting physiologic defects in patients with CF.

L5 ANSWER 37 OF 40 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on
STN DUPLICATE 21
AN 1992:34170 BIOSIS
DN PREV199293023445; BA93:23445
TI SYNTHESIS AND EXPRESSION IN ESCHERICHIA-COLI OF A GENE FOR KAPPA
BUNGAROTOXIN.
AU FIORDALISI J J [Reprint author]; FETTER C H; TENHARMSEL A; GIGOWSKI R;
CHIAPPINELLI V A; GRANT G A
CS DEP BIOCHEMISTRY MOLECULAR BIOPHYSICS, WASHINGTON UNIV SCHOOL MED, ST
LOUIS, MISSOURI 63110, USA
SO Biochemistry, (1991) Vol. 30, No. 42, pp. 10337-10343.
CODEN: BICHAW. ISSN: 0006-2960.
DT Article
FS BA
LA ENGLISH
ED Entered STN: 6 Jan 1992
Last Updated on STN: 6 Mar 1992
AB A gene which codes for the 66-residue polypeptide of .kappa.-bungarotoxin has been chemically synthesized by linking together 3 synthetic double-stranded oligonucleotides in a bacterial plasmid. The synthesis incoporated six unique silent restriction sites spaced throughout the gene for use in cassette mutagenesis. Direct expresssion of the .kappa.-bungarotoxin polypeptide by itself in Escherichia coli failed to result with rat ***intestinal*** ***fatty*** ***acid*** ***binding*** ***protein*** under control of the nalidixic acid inducible recA ***promoter*** . Two fusion protein constructs were prepared that differed only in the cleavage site between the fatty acid binding protein and the toxin polypeptide. One contained a factor Xa cleavage site, and the other, since the toxin itself is devoid of methionine, contained a methionyl residue that served as a cyanogen bromide cleavage site. The fusion proteins were isolated by ion-exchange chromatography and reverse-phase HPLC. The construct containing the factor Xa cleavage site could not be cleaved under nondenaturing conditions. On the other hand, .kappa.-bungarotoxin was efficiently cleaved from the methionyl fusion protein with CNBr. The toxin polypeptide was isolated by reverse-phase HPLC and ion-exchange chromatography and produced a complete and specific blockade of neuronal nicotinic acetylcholine receptors in chick ciliray ganglia which was indistinguishable from that produced by a comparable amount of venom-purified .kappa.-bungarotoxin.

L5 ANSWER 38 OF 40 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on
STN
AN 1991:356403 BIOSIS
DN PREV199141040918; BR41:40918
TI FUNCTIONAL ANALYSIS OF THE ***INTESTINAL*** ***FATTY*** ***ACID*** ***BINDING*** ***PROTEIN*** GENE ***PROMOTER*** IN TRANSGENIC MICE.
AU COHN S M [Reprint author]; SIMON T C; ROTH K A; BIRKENMEIER E H; GORDON J
I
CS WASHINGTON UNIV SCH MED, ST LOUIS, MO, USA
SO Clinical Research, (1991) Vol. 39, No. 2, pp. 222A.
Meeting Info.: JOINT MEETING OF THE ASSOCIATION OF AMERICAN PHYSICIANS,
THE AMERICAN SOCIETY FOR CLINICAL INVESTIGATION, AND THE AMERICAN
FEDERATION FOR CLINICAL RESEARCH, SEATTLE, WASHINGTON, USA, MAY 3-6, 1991.
CLIN RES.
CODEN: CLREAS. ISSN: 0009-9279.
DT Conference; (Meeting)
FS BR
LA ENGLISH
ED Entered STN: 1 Aug 1991
Last Updated on STN: 1 Aug 1991

L5 ANSWER 39 OF 40 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED.

on STN DUPLICATE 22
AN 90315130 EMBASE
DN 1990315130
TI Expression of rat ***intestinal*** ***fatty*** ***acid*** ***binding*** ***protein*** in E. coli and its subsequent structural analysis: A model system for studying the molecular details of fatty acid-protein interaction.
AU Sacchettini J.C.; Banaszak L.J.; Gordon J.I.
CS Dept. Biochem. Mol. Biophysics, Washington Univ. Sch. Med., Box 8231, 660 S. Euclid Ave.,St. Louis, MO 63110, United States
SO Molecular and Cellular Biochemistry, (1990) 98/1-2 (81-93).
ISSN: 0300-8177 CODEN: MCBIB8
CY Netherlands
DT Journal; Conference Article
FS 004 Microbiology
029 Clinical Biochemistry
LA English
SL English
AB A prokaryotic expression vector containing the rec A ***promoter*** and a translational enhancer element from the gene 10 leader of bacteriophage T7 was used to direct efficient synthesis of rat ***intestinal*** ***fatty*** ***acid*** ***binding*** ***protein*** (I-FABP) in E. coli. Expression of I-FABP in E. coli has no apparent, deleterious effects on the organism. High levels of expression of I-FABP mRNA in supE+ strains of E. coli, such as JM101, is associated with suppression of termination at its UGA stop codon. This can be eliminated by using a supE- strain as MG 1655 and by site-directed mutagenesis of the cDNA to create an in frame UAA stop codon E. coli-derived rat I-FABP lacks its initiator Met residues. It has been crystallized with and without bound palmitate. High resolution x-ray crystallographic studies of the 131 residue apo- and holo-proteins have revealed the following. I-FABP contains 10 anti-parallel .beta.-strands organized into two orthogonally situated .beta.-sheets. The overall conformation of the protein resembles that of a clam - hence the term .beta.-clam. The bound ligand is located in the interior of the protein. Its carboxylate group forms part of a unique five member hydrogen bonding network consisting of two ordered solvent molecules as well as the side chains of Arg106 and Gln115. Thy hydrocarbon chain of the bound C16:0 fatty acid has a distinctive bent conformation with a slight left-handed helical twist. This conformation is maintained by interactions with the side chains of a number of hydrophobic and aromatic amino acids. Apo-I-FABP has a similar overall conformation to holo-I-FABP indicating that the .beta.-clam structure is stable even without bound ligand. The space occupied by bound ligand in the core of the holo-protein is occupied by additional ordered solvent molecules in the apo-protein. Differences in the side chain orientations of several residues located over a potential opening to the cores of the apo- and holo-proteins suggest that solvent may play an important role in the binding mechanism. Comparison of the C.alpha. coordinates of apo- and holo-I-FABP with those of other proteins indicates it is a member of a superfamily that currently includes (i) 10 mammalian intracellular lipid binding proteins, (ii) the photoactive yellow protein from the purple photoautotrophic bacterium Ectothiorhodospira halophila and (iii) a group of extracellular lipid binding proteins from a diverse number of phyla that have a common .beta.'barrel' consisting of 8 anti-parallel .beta.-strands stacked in two nearly orthogonal sheets. In summary, E. coli-derived I-FABP not only represents a useful model for assessing the atomic details of fatty acid-protein interactions and the mechanisms which regulate acquisition and release of this type of ligand, but also structure/function relationships in other superfamily members.

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TI TRANSGENIC MICE CONTAINING ***INTESTINAL*** ***FATTY*** ***ACID*** - ***BINDING*** ***PROTEIN*** -HUMAN GROWTH HORMONE
FUSION GENES EXHIBIT CORRECT REGIONAL AND CELL-SPECIFIC EXPRESSION OF THE
REPORTER GENE IN THEIR SMALL INTESTINE.
AU SWEETSER D A [Reprint author]; HAUFT S M; HOPPE P C; BIRKENMEIER E H;
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AB The rat ***intestinal*** ***fatty*** ***acid*** ***binding*** ***protein*** (I-FABP) gene exhibits cell-specific as well as regional differences in its expression within the continuously regenerating small intestinal epithelium. To investigate the underlying mechanisms, we linked portions of its 5' nontranscribed domain to the human growth hormone (hGH) gene and analyzed expression of the hGH reporter in transgenic mice by RNA blot, solution hybridization, and immunocytochemical techniques. Sequences located within 277 nucleotides of the start site of I-FABP transcription are sufficient to limit hGH expression to the intestine. Although the absolute levels of hGH mRNA in the duodenum and proximal jejunum of these transgenic mice were similar to those of I-FABP mRNA, steady-state hGH mRNA concentrations were .apprxeq. 100 times lower in their distal small intestine. Addition of nucleotides .sbd.278 to .sbd.1178 of the I-FABP gene "restored" hGH mRNA concentrations in the distal jejunum and ileum to levels comparable to murine I-FABP mRNA. Serum hGH levels were 1000 times lower in the "short ***promoter*** " transgenic mice compared to animals with the "long ***promoter*** " transgene, indicating that efficient distal small intestinal hGH expression is required to produce elevated hGH concentrations in serum. The distribution of hGH in villus-associated enterocytes and goblet cells and its lack of expression in the crypts of Lieberkuhn mimicked that of the endogenous I-FABP gene product in all transgenic pedigrees. However, bands of hGH-negative cells extending from the base to the tips of villi were frequently observed in mice that were heterozygous for the short ***promoter*** transgene. This mosaic staining was not observed for I-FABP. These data suggest that (i) different cis-acting sequences may be required for complete expression of proximal.sbd.distal I-FABP gradients than for recapitulation of its normal crypt.sbd.villus tip distribution; (ii) differences may exist in the export pathways of secreted proteins within enterocytes located in various regions of the small intestine; and (iii) there may be subtle genetic differences among various crypt stem cells that can be detected in vivo by observing mosaic patterns of transgene expression along the villus epithelium.

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